

Letters to the Editor

High alcohol intake and slow progression to AIDS

It is not yet understood why some HIV-1 infected persons develop AIDS within a few years after infection and others remain healthy and with a normal CD4 cell count for many years. In order to assess the influence of different epidemiological and behavioural factors and the role of infection with pathogens which might act as co-factors for disease progression in HIV-1 infected people, a multicentre study was started in Madrid in September 1992.

Rapid progressors (RP) and slow progressors (SP) were checked in a group of 1783 HIV-infected persons regularly attending three medical centres in Madrid. Definition criteria were for RP: infection occurred in the last 5 years and with a current CD4 + count repeatedly below 200/mm³; and for SP: more than 8 years of confirmed HIV-1 infection and with the number of CD4 + cells consistently above 500/mm³ in the absence of any antiretroviral therapy and without symptoms. One hundred persons (5.6%) met the criteria for SP and 12 (0.7%) for RP. Of 48 SP with a CD4 count monitored for more than 5 years, 16 (33%) were absolute non-progressors, maintaining a normal and stable CD4 + count.

Variables more frequently recognised in the SP group compared with the RP group were: previous injecting drug addiction (IDA) practices ($p = 0.0002$), low cultural level ($p = 0.0023$), younger age beginning high-risk practices ($p = 0.0039$), male gender ($p = 0.0370$) and prolonged high alcohol intake ($p = 0.0391$), defined as consumption of alcohol above 100 g daily for more than 3 years. Co-infection with hepatitis B and C viruses or other infectious agents which could act as co-factors was not seen more frequently in RP. Categorising by the route of infection (sexually or parenterally), a younger age beginning high-risk practices was associated with SP in injecting drug users, and female gender was associated with RP in people infected through sexual contact. Chronic high alcohol intake showed a strong association with SP amongst injected drug users although it did not achieve significance ($p = 0.0995$). There was no evidence linking this effect to any particular drink or liquor. Nevertheless, in three HIV-positive heavy drinkers with SP, an enhancement of plasma HIV-RNA was not seen two weeks after stopping the intake of alcohol, or a fall after resuming alcohol consumption.

Although there have been reports of rapid progression to AIDS in alcoholic HIV-infected patients,¹ longitudinal studies in large cohorts have not been able to find any association between high alcohol intake and worse prognosis in HIV-infected patients.^{2,3} We postulate that in HIV-infected subjects with preserved immune status, chronic high alcohol intake could have a protective effect against CD4 + depletion, as has recently been proposed for two other immunosuppressive substances, as corticosteroids⁴ and cyclosporin A.⁵ Two main reasons could explain this unexpected beneficial effect of alcohol. First, some substances

present in many alcoholic drinks, as flavonoids in red wine, have a powerful antioxidant activity,⁶ which can reduce virus expression in infected cells. Second, ethanol can suppress the activation of lymphocytes and monocytes/macrophages, which function as an important reservoir for the virus. Indirectly, expression of the virus in infected cells could be suppressed, causing a decline in HIV viraemia and perhaps yielding a prolonged survival in these patients. Of course, before recommending a couple of whiskies daily, doctors and patients should be aware that this hypothetical benefit of alcohol on HIV replication needs to be balanced with other disadvantageous effects of alcoholism, mainly on liver function and nutritional status, which by other means might cause immune dysfunction.

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- 1 Fong I, Read S, Wainberg M, Chia W, Major C. Alcoholism and rapid progression to AIDS after seroconversion. *Clin Infect Dis* 1994;19:337-8.
- 2 Kaslow R, Blackwelder W, Ostrow D. No evidence for a role of alcohol or other psychoactive drugs in accelerating immunodeficiency in HIV-1-positive individuals. A report from the Multicenter AIDS Cohort Study. *JAMA* 1989;261:3424-9.
- 3 Coates R, Farewell V, Raboud J, Reed S, MacFadden D, Calzavara L. Cofactors of progression to AIDS in a cohort of male sexual contacts of men with HIV disease. *Am J Epidemiol* 1990;132:717-22.
- 4 Andrieu J, Lu W, Levy R. Sustained increases in CD4 cell counts in asymptomatic HIV type 1 seropositive patients treated with prednisolone for one year. *J Infect Dis* 1995;171:523-7.
- 5 Weber J, Galpin S. Cyclosporin A. *Nature* 1995;375:198.
- 6 Maxwell S, Cruickshank A, Thorpe G. Red wine and antioxidant activity in serum. *Lancet* 1994;344:193-4.

Dietary intervention in HIV: a comparison of patients receiving oral, enteral and parenteral nutrition

Weight loss is an important complication of human immunodeficiency virus (HIV) infection. Nutritional deficiency contributes to the progression of disease and susceptibility to opportunistic infections and weight loss is a major factor associated with the time of death.¹⁻³ Weight loss associated with HIV is a complex process and may result from decreased nutrient intake, impaired nutrient absorption or increased nutritional requirements. The optimum method of delivering nutritional support at each stage of infection has not been established. We therefore reviewed patients referred to a designated HIV dietician over a two year period and compared the characteristics and outcome of patients receiving dietary advice only, oral supplementation, enteral feeding and parenteral nutrition.

Information on all adult patients seen by the HIV dietician over a two year period during 1993-95 was obtained from the medical and